Search Pgm 8

WEST Search History

DATE: Tuesday, August 05, 2003

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DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L4	L3 and @ay<2000	48	L4
L3	L2 same (tissue or cell)	100	L3
L2	L1 same (identif$ or compar$)	166	L2
L1	(methylation or methylated) near2 pattern	1387	L1

END OF SEARCH HISTORY

Search Paper 6

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NEWS 17	May 15	MEDLINE file segment of TOXCENTER reloaded
NEWS 18	May 15	Supporter information for ENCOMPPAT and ENCOMPLIT updated
NEWS 19	May 19	Simultaneous left and right truncation added to WSCA
NEWS 20	May 19	RAPRA enhanced with new search field, simultaneous left and right truncation
NEWS 21	Jun 06	Simultaneous left and right truncation added to CBNB
NEWS 22	Jun 06	PASCAL enhanced with additional data
NEWS 23	Jun 20	2003 edition of the FSTA Thesaurus is now available
NEWS 24	Jun 25	HSDB has been reloaded
NEWS 25	Jul 16	Data from 1960-1976 added to RDISCLOSURE
NEWS 26	Jul 21	Identification of STN records implemented
NEWS 27	Jul 21	Polymer class term count added to REGISTRY
NEWS 28	Jul 22	INPADOC: Basic index (/BI) enhanced; Simultaneous Left and Right Truncation available

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FILE 'HOME' ENTERED AT 10:26:02 ON 05 AUG 2003

=> file .pub
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

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FILE 'BIOSIS' ENTERED AT 10:26:15 ON 05 AUG 2003
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=> s (methylation or methylated) (2A) pattern
L1         3453 (METHYLATION OR METHYLATED) (2A) PATTERN

=> s l1 and (identif or compar)
) IS NOT A RECOGNIZED COMMAND
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=> s l1 and (identif or compar)
) IS NOT A RECOGNIZED COMMAND
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For a list of commands available to you in the current file, enter
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=> s l1 and (identif? or compar?)
L2         1131 L1 AND (IDENTIF? OR COMPAR?)

=> s l1 and ((identif? or compar?) (3A) (tissue or cell))
L3          136 L1 AND ((IDENTIF? OR COMPAR?) (3A) (TISSUE OR CELL))

=> duplicate remove l3
DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L3
L4            87 DUPLICATE REMOVE L3 (49 DUPLICATES REMOVED)

=> d 1-10 bib ab

L4   ANSWER 1 OF 87     MEDLINE on STN                  DUPLICATE 1
AN   2003243810     MEDLINE
DN   22651093   PubMed ID: 12651856
TI   Characterization of regulatory elements and methylation
     pattern of the autoimmune regulator (AIRE) promoter.
AU   Murumagi Astrid; Vahamurto Perttu; Peterson Part
CS   Institute of Medical Technology, University of Tampere and Department of
     Pathology, Tampere University Hospital, Lenkkeilijankatu 6, Finland.
SO   JOURNAL OF BIOLOGICAL CHEMISTRY, (2003 May 30) 278 (22) 19784-90.
     Journal code: 2985121R. ISSN: 0021-9258.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   200307
ED   Entered STN: 20030528
     Last Updated on STN: 20030711
     Entered Medline: 20030710
AB   Defects in the AIRE gene cause a monogenic autoimmune syndrome APECED
     (autoimmune polyendocrinopathy candidiasis ectodermal dystrophy), which is
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characterized by loss of self-tolerance to multiple organs. In concordance with its role in immune tolerance, AIRE is most strongly expressed in thymic epithelial cells and in cells of monocytic-dendritic lineage. The AIRE protein has been shown to function as a transcriptional regulator, however, the mechanisms regulating AIRE gene expression are not known. Here we have characterized the AIRE promoter region by identifying a minimal promoter region within 350 bp of the translation initiation codon. Electrophoretic mobility shift assays and transient transfections with mutated promoter constructs revealed a functional TATA box (-163 to -153) and binding sites for transcription complexes AP-1 (-307 to -296), NF-Y (-213 to -202), and Sp1 (-202 to -189). The presence of a 390-bp CpG island within the proximal promoter suggested that cytosine methylation has a role in transcriptional regulation of AIRE, which was supported by in vitro methylation experiments of promoter constructs. Sodium bisulfite sequencing showed a less methylated status of AIRE promoter in the thymic epithelial **cell** line TEC1A3 **compared** with HeLa and monocytic cells U937 and THP-1. Real-time PCR analysis showed that treatment with 5-aza-2'-deoxycytidine (5-azaCdR), a DNA methyltransferase inhibitor, up-regulated AIRE transcript levels in TEC1A3, U937, and HeLa cells and that even greater activations in TEC1A3 and U937 cells were observed using combined treatments with deacetylase inhibitor trichostatin A. These results suggest that AIRE gene expression is modulated through modifications in chromatin methylation and acetylation.

L4 ANSWER 2 OF 87 MEDLINE on STN
AN 2003341672 IN-PROCESS
DN 22756060 PubMed ID: 12874021
TI Frequent hypomethylation of multiple genes overexpressed in pancreatic ductal adenocarcinoma.
AU Sato Norihiro; Maitra Anirban; Fukushima Noriyoshi; van Heek N Tjarda; Matsubayashi Hiroyuki; Iacobuzio-Donahue Christine A; Rosty Christophe; Goggins Michael
CS Department of Pathology, The Johns Hopkins Medical Institutions, Baltimore, Maryland 21205, USA.
NC CA62924 (NCI)
CA90709 (NCI)
SO CANCER RESEARCH, (2003 Jul 15) 63 (14) 4158-66.
Journal code: 2984705R. ISSN: 0008-5472.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20030723
Last Updated on STN: 20030731
AB To investigate the relationship between DNA hypomethylation and gene overexpression in pancreatic cancer, we analyzed the methylation status of a subset of 18 genes previously identified by global gene expression studies as overexpressed in pancreatic cancer **tissues** **compared** with normal pancreas. For comparison, we determined the methylation status of 14 genes not known to be overexpressed in pancreatic cancer. Methylation-specific PCR analysis revealed that 19 of these 32 genes were methylated at their 5' CpGs in normal pancreas. We then analyzed these 19 genes for their **methylation pattern** in pancreatic cancers and found that all 7 of the genes (claudin4, lipocalin2, 14-3-3sigma, trefoil factor2, S100A4, mesothelin, and prostate stem cell antigen) that were overexpressed in the neoplastic cells of pancreatic cancers and not expressed in normal pancreatic duct displayed a high prevalence of hypomethylation in pancreatic cancer cell lines and primary pancreatic carcinomas. By contrast, only 1 of 12 genes not overexpressed in pancreatic cancer demonstrated hypomethylation (P = 0.0002). In pancreatic cancer cell lines that retained methylation of 1 or more of the 7 aforementioned overexpressed and hypomethylated genes, treatment with 5-aza-2'-deoxycytidine or with trichostatin A, either alone or in combination, almost invariably reactivated the transcription of each

of these 7 genes. These results indicate that gene hypomethylation is a frequent epigenetic event in pancreatic cancer and is commonly associated with the overexpression of affected genes.

L4 ANSWER 3 OF 87 MEDLINE on STN DUPLICATE 2
AN 2003124235 MEDLINE
DN 22516403 PubMed ID: 12629412
TI The roles of supernumerical X chromosomes and XIST expression in testicular germ cell tumors.
AU Kawakami Takahiro; Okamoto Keisei; Sugihara Hiroyuki; Hattori Takanori; Reeve Anthony E; Ogawa Osamu; Okada Yusaku
CS Department of Urology, Shiga University of Medical Science, Otsu, Japan.
SO JOURNAL OF UROLOGY, (2003 Apr) 169 (4) 1546-52.
Journal code: 0376374. ISSN: 0022-5347.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200304
ED Entered STN: 20030318
Last Updated on STN: 20030403
Entered Medline: 20030402
AB PURPOSE: An overabundance of X chromosomes in testicular germ **cell** tumors and the **identification** of the candidate testicular germ cell tumor susceptibility gene TGCT1 on Xq27 highlight the potential involvement of X chromosomes in testicular germ cell tumor pathogenesis. The current study was designed to shed light on the question whether the multiple X chromosomes in testicular germ cell tumor are active or inactive through a complex mechanism of X chromosomal gain and XIST expression. MATERIALS AND METHODS: We analyzed 4 testicular germ cell tumor derived cell lines and 20 primary testicular germ cell tumor tissues. The number of X chromosomes was determined by fluorescence in situ hybridization using the X chromosome specific probe. The expression patterns of XIST and the 3 X-linked genes androgen receptor (AR), fragile X mental retardation (FMR1) and Glypican 3 (GPC3) were studied by reverse transcriptase-polymerase chain reaction. Bisulfite genomic sequencing was used to analyze the **methylation patterns** of the AR, FMR1 and GPC3 genes. The relative expression levels of the 2 X-linked proto-oncogenes ARAF1 and ELK1 were assayed by quantitative reverse transcriptase-polymerase chain reaction. RESULTS: XIST expression was common in seminomatous testicular germ cell tumors (2 of 2 or 100% of seminoma derived cell lines and 10 of 12 or 83% of seminomatous testicular germ cell tumor tissues) but not in nonseminomatous testicular germ cell tumors (0 of 2 or 0% nonseminoma derived cell lines and 2 of 8 or 25% of nonseminomatous testicular germ cell tumor tissues). However, X chromosomal gain was consistently observed in the 2 types of tumors. XIST expression in testicular germ cell tumors and normal testicular parenchyma was not associated with methylation of the AR, FMR1 or GPC3 genes. After determining the expression patterns of AR, FMR1 and GPC3 in testicular germ cell tumor samples we concluded that multiple X chromosomes in testicular germ cell tumors were predominantly hypomethylated and active regardless of XIST expression. The biological significance of excess active X chromosomes in testicular germ cell tumors was suggested by enhanced expression of the 2 X-linked oncogenes ARAF1 and ELK1 in the testicular germ cell tumor derived cell lines. CONCLUSIONS: The current data may suggest the potential oncogenic implications of X chromosomal gain in testicular germ cell tumors.

L4 ANSWER 4 OF 87 MEDLINE on STN
AN 2003262579 MEDLINE
DN 22672756 PubMed ID: 12788407
TI Relevance of DNA methylation in the management of cancer.
AU Esteller Manel
CS Cancer Epigenetics Laboratory at the Spanish National Cancer Centre

(CNiO), Madrid, Spain. mesteller@cnio.es. <mesteller@cnio.es>
SO Lancet Oncol, (2003 Jun) 4 (6) 351-8. Ref: 76
Journal code: 100957246. ISSN: 1470-2045.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200306
ED Entered STN: 20030606
Last Updated on STN: 20030628
Entered Medline: 20030627
AB Many genetic and environmental factors contribute to development of cancer, but DNA methylation may provide a link between these influences. Genome stability and normal gene expression are largely maintained by a fixed and predetermined **pattern** of DNA **methylation**.
In cancer, this idealistic scenario is disrupted by an interesting phenomenon: the hypermethylation of regulatory regions called CpG islands in some tumour suppressor genes--eg, BRCA1, hMLH1, p16INK4a, APC, VHL--which causes their inactivation. Development of new techniques that couple bisulphite modification with PCR has enabled these alterations to be studied in all types of biological fluids and archived tissues. Potentially, there are four types of translational studies that can be used to investigate the aberrant **pattern** of DNA **methylation** in cancer. First, CpG island hypermethylation can be used as a marker to **identify** cancer **cells** from biological samples, eg, serum and urine. This technique is highly sensitive and informative because profiles of tumour-suppressor-gene inactivation are specific to particular cancers. Second, single and combined genes that are inactivated by promoter hypermethylation, such as p16INK4a and DAPK, can be used as prognostic factors. Third, products of genes that are silenced by DNA methylation can be used as biomarkers of response to chemotherapy or hormone therapy--eg, the DNA repair O6-methylguanine-DNA methyltransferase and the oestrogen receptor. Finally, dormant tumour suppressor genes can be reactivated by DNA demethylating drugs, with the aim of reversing the neoplastic phenotype. These are new avenues worth exploring in the fight against cancer.

L4 ANSWER 5 OF 87 MEDLINE on STN DUPLICATE 3
AN 2003056780 MEDLINE
DN 22454726 PubMed ID: 12566254
TI X-inactivation patterns in human embryonic and extra-embryonic tissues.
AU Zeng S-M; Yankowitz J
CS Department of Obstetrics and Gynecology, University of Iowa College of Medicine, Iowa City 52242, USA.
SO PLACENTA, (2003 Feb-Mar) 24 (2-3) 270-5.
Journal code: 8006349. ISSN: 0143-4004.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200307
ED Entered STN: 20030205
Last Updated on STN: 20030729
Entered Medline: 20030728
AB Mice have skewed X chromosome inactivation (XCI) in extraembryonic tissue while examination of human placentae have yielded conflicting results. We investigated XCI patterns in human embryonic and extra-embryonic tissues. First and early second trimester placental and foetal tissues were collected. Cytotrophoblasts were isolated from the placentae. Female samples were identified and X-inactivation patterns were determined by analysis of androgen receptor (HAR) **methylation patterns**. Among 55 females heterozygous at the HAR, 37 had random and 18 skewed

XCI. In foetal tissues a skewed XCI pattern was only observed in one liver and one intestine sample. A greater incidence of skewed XCI pattern was present in extra-embryonic **compared** to embryonic **tissues** (P=0.022). A markedly skewed XCI pattern was only found in one cytotrophoblast sample. Random and skewed XCI patterns were detected in human embryonic and extra-embryonic tissues. The extra-embryonic tissue had a higher proportion of skewed XCI, but marked skewed XCI was uncommon in both tissues. Skewed XCI may not play a role in normal human placentation.

L4 ANSWER 6 OF 87 MEDLINE on STN DUPLICATE 4
AN 2002271111 MEDLINE
DN 22005988 PubMed ID: 12010815
TI Down-regulation of candidate tumor suppressor genes within chromosome band 13q14.3 is independent of the DNA **methylation pattern** in B-cell chronic lymphocytic leukemia.
AU Mertens Daniel; Wolf Stephan; Schroeter Petra; Schaffner Claudia; Dohner Hartmut; Stilgenbauer Stephan; Lichter Peter
CS Abteilung "Organisation komplexer Genome," Deutsches Krebsforschungszentrum, Heidelberg, Germany.
SO BLOOD, (2002 Jun 1) 99 (11) 4116-21.
Journal code: 7603509. ISSN: 0006-4971.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200207
ED Entered STN: 20020516
Last Updated on STN: 20020702
Entered Medline: 20020701
AB Loss of genomic material from chromosomal band 13q14.3 is the most common genetic imbalance in B-cell chronic lymphocytic leukemia (B-CLL) and mantle cell lymphoma, pointing to the involvement of this region in a tumor suppressor mechanism. From the minimally deleted region, 3 candidate genes have been isolated, RFP2, BCMS, and BCMSUN. DNA sequence analyses have failed to detect small mutations in any of these genes, suggesting a different pathomechanism, most likely haploinsufficiency. We, therefore, tested B-CLL patients for epigenetic aberrations by measuring expression of genes from 13q14.3 and methylation of their promotor region. RB1, CLLD7, KPNA3, CLLD6, and RFP2 were down-regulated in B-CLL patients as **compared** with B **cells** of healthy donors, with RFP2 showing the most pronounced loss of expression. To test whether this loss of gene expression is associated with methylation of CpG islands in the respective promotor regions, we performed methylation-sensitive quantitative polymerase chain reaction analyses and bisulfite sequencing on DNA from B-CLL patients. No difference in the **methylation patterns** could be detected in any CpG island of the minimally deleted region. Down-regulation of genes within chromosomal band 13q14.3 in B-CLL is in line with the concept of haploinsufficiency, but this tumor-specific phenomenon is not associated with DNA methylation.

L4 ANSWER 7 OF 87 MEDLINE on STN
AN 2003201138 MEDLINE
DN 22606520 PubMed ID: 12722382
TI [Estimation of DNA methylation level in nonendometrial uterus malignancies].
Ocena poziomu metylacji DNA w tkankach zlosliwych nowotworow nienablonkowych trzonu macicy.
AU Popiela Andrzej; Gabrys Marian Stanislaw; Rabczynski Jerzy; Panszczyk Marcin; Keith Gerard; Baranowski Wlodzimierz
CS II Katedry i Kliniki Ginekologii AM we Wroclawiu.
SO GINEKOLOGIA POLSKA, (2002 Nov) 73 (11) 962-5.
Journal code: 0374641. ISSN: 0017-0011.

CY Poland
DT Journal; Article; (JOURNAL ARTICLE)
LA Polish
FS Priority Journals
EM 200306
ED Entered STN: 20030501
Last Updated on STN: 20030603
Entered Medline: 20030602
AB OBJECTIVES: Rebuilding of genome structure leads to many pathological states including neoplastic malignancies. Rebuilding often occurs as a process caused by disturbances in gene silencing mechanism. DNA **methylation pattern** is one of the most important mechanisms connected to gene's silencing. Estimation of DNA methylation level in nonendometrial uterine neoplastic **tissues compared** to normal endometrial samples was the aim of this study. DESIGN: It was to be shown, that changes in methylation rate in promotory regions could lead to carcinogenesis in particular cell. Authors describe an analysis of DNA methylation level in nonendometrial neoplastic uterine **tissues compared** to DNA methylation level in normal endometrium. MATERIALS AND METHODS: Tissue samples from 9 women with tumor mixtus mesodermalis were collected. 12 samples were normal endometrium-control group. DNA was isolated from tissues and than we performed an estimation of DNA methylation levels. Than we performed a statistical analysis of results. RESULTS: The median DNA methylation level was significantly higher in neoplastic tissues than in normal endometrium. CONCLUSIONS: Authors conclude, that DNA methylation level is higher in neoplastic tissues, but does not correlate with clinical stage of the disease.

L4 ANSWER 8 OF 87 MEDLINE on STN
AN 2002307425 MEDLINE
DN 22044148 PubMed ID: 12049211
TI Epigenetic mechanisms for primary differentiation in mammalian embryos.
AU Patkin Eugene L
CS Department of Molecular Genetics, Institute of Experimental Medicine, Russian Academy of Medical Sciences, St Petersburg.
SO INTERNATIONAL REVIEW OF CYTOLOGY, (2002) 216 81-129. Ref: 297
Journal code: 2985180R. ISSN: 0074-7696.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200212
ED Entered STN: 20020611
Last Updated on STN: 20021217
Entered Medline: 20021204
AB This review examines main developments related to the interface between primary mammalian cell differentiation and various aspects of chromosomal structure changes, such as heterochromatin dynamics, DNA methylation, mitotic recombination, and inter- and intrachromosomal differentiation. In particular, X chromosome difference, imprinting, chromosomal banding, **methylation pattern**, single-strand DNA breaks, sister chromatid exchanges (SCEs), and sister chromatid asymmetry are considered. A hypothesis is put forward which implies the existence of an epigenetic asymmetry versus mirror symmetry of sister chromatids for any DNA sequences. Such epigenetic asymmetry appears as a result of asymmetry of sister chromatid organization and of SCE and is a necessary (not sufficient) condition for creating cell diversity. The sister chromatid asymmetry arises as a result of consecutive rounds of active and passive demethylation which leads after chromatin assembly events to chromatid difference. Single-strand DNA breaks that emerge during demethylation trigger reparation machinery, provend as sister chromatid exchanges, which

are not epigenetically neutral in this case. Taken together, chromatid asymmetry and SCE lead to cell diversity regarding their future fate. Such cells are considered pluripotent stem cells which after interplay between a set of chromosomal domains and certain substances localized within the cytoplasmic **compartments** (and possibly **cell** interactions) can cause sister cells to express different gene chains. A model is suggested that may be useful for stem cell technology and studies of carcinogenesis.

L4 ANSWER 9 OF 87 MEDLINE on STN
AN 2002126524 MEDLINE
DN 21851320 PubMed ID: 11861926
TI Tumour class prediction and discovery by microarray-based DNA methylation analysis.
AU Adorjan Peter; Distler Jurgen; Lipscher Evelyne; Model Fabian; Muller Jurgen; Pelet Cecile; Braun Aron; Florl Andrea R; Gutig David; Grabs Gabi; Howe Andre; Kursar Mischo; Lesche Ralf; Leu Erik; Lewin Andre; Maier Sabine; Muller Volker; Otto Thomas; Scholz Christian; Schulz Wolfgang A; Seifert Hans-Helge; Schwope Ina; Ziebarth Heike; Berlin Kurt; Piepenbrock Christian; Olek Alexander
CS Information Sciences, Biomedical Research and Development, Epigenomics AG, Berlin, Germany.
SO NUCLEIC ACIDS RESEARCH, (2002 Mar 1) 30 (5) e21.
Journal code: 0411011. ISSN: 1362-4962.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200203
ED Entered STN: 20020226
Last Updated on STN: 20020312
Entered Medline: 20020311
AB Aberrant DNA methylation of CpG sites is among the earliest and most frequent alterations in cancer. Several studies suggest that aberrant methylation occurs in a tumour type-specific manner. However, large-scale analysis of candidate genes has so far been hampered by the lack of high throughput assays for methylation detection. We have developed the first microarray-based technique which allows genome-wide assessment of selected CpG dinucleotides as well as quantification of methylation at each site. Several hundred CpG sites were screened in 76 samples from four different human tumour types and corresponding healthy controls. Discriminative CpG dinucleotides were **identified** for different **tissue** type distinctions and used to predict the tumour class of as yet unknown samples with high accuracy using machine learning techniques. Some CpG dinucleotides correlate with progression to malignancy, whereas others are methylated in a tissue-specific manner independent of malignancy. Our results demonstrate that genome-wide analysis of **methylation patterns** combined with supervised and unsupervised machine learning techniques constitute a powerful novel tool to classify human cancers.

L4 ANSWER 10 OF 87 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:336069 BIOSIS
DN PREV200300336069
TI Aberrant DNA Methylation in Chronic Lymphocytic Leukemia: A Role in Pathogenesis.
AU Raval, Aparna (1); Rush, Laura J. (1); Funchain, Pauline (1); Flak, Erin (1); Davis, Melanie (1); Johnson, Amy J. (1); Byrd, John C. (1); Plass, Christoph (1)
CS (1) Division of Human Cancer Genetics, Ohio State University, Columbus, OH, USA USA
SO Blood, (November 16 2002) Vol. 100, No. 11 , pp. Abstract No. 1469. print.
Meeting Info.: 44th Annual Meeting of the American Society of Hematology Philadelphia, PA, USA December 06-10, 2002 American Society of Hematology

. ISSN: 0006-4971.
DT Conference
LA English
AB Chronic lymphocytic leukemia (CLL) is the most common leukemia in the Western world. Identifying genetic defects involved in CLL remains a high priority. Interphase cytogenetic studies demonstrate loss of chromosomal material in up to 80% of CLL patients but the affected genes have yet to be identified. Epigenetic changes, such as aberrant promoter methylation, are another pathway by which loss of gene function can occur. The contribution of DNA methylation in CLL has not been studied on a genome wide level. Here we report the first genome wide scan for aberrant DNA methylation in CLL samples using Restriction Landmark Genomic Scanning (RLGS). RLGS is a highly reproducible two-dimensional gel electrophoresis that allows the determination of the methylation status of up to 2000 promoter sequences in a single gel using a methylation-sensitive restriction enzyme. In the present study we sought to determine if global **methylation patterns** in CLL patients were different than that observed in normal B-cells. RLGS analysis examined 10 CLL patients with comparison to CD19 selected lymphocytes from normal donors using two methylation-sensitive restriction enzymes Not I and Asc I allowing assessment of 3600 CpG islands in the human genome. Parallel control comparison to neutrophils derived from each CLL patient allowed elimination of polymorphisms that could mimic sites of methylation. We found a marked variation in the amount of aberrant methylation in patient samples ranging from 1-6% as **compared** to normal B **cells** . Cloning of more than 50 methylated loci has been performed utilizing NotI-EcoRV and AscI-EcoRV libraries, which identified different genes and EST sequences. Promoter methylation of selected genes was confirmed by bisulfite sequencing and Southern blot. The expression analysis as well as clinical and interphase cytogenetic correlations may provide additional insight into molecular pathogenesis of CLL and will be presented.

=> d his

(FILE 'HOME' ENTERED AT 10:26:02 ON 05 AUG 2003)

FILE 'MEDLINE, BIOSIS' ENTERED AT 10:26:15 ON 05 AUG 2003
L1 3453 S (METHYLATION OR METHYLATED) (2A) PATTERN
L2 1131 S L1 AND (IDENTIF? OR COMPAR?)
L3 136 S L1 AND ((IDENTIF? OR COMPAR?) (3A) (TISSUE OR CELL))
L4 87 DUPLICATE REMOVE L3 (49 DUPLICATES REMOVED)

=> s l4 and py<2000
L5 64 L4 AND PY<2000

=> d 1-10 bib ab

L5 ANSWER 1 OF 64 MEDLINE on STN
AN 2002049328 MEDLINE
DN 21633181 PubMed ID: 11776622
TI The role of cytokeratin 13 gene in human nasopharyngeal carcinoma.
AU Qiu Y; Tian Y; Xiao J
CS Department of Otolaryngology, Xiang Ya Hospital, Hunan Medical University, Changsha 410008.
SO CHUNG-HUA CHUNG LIU TSA CHIH [CHINESE JOURNAL OF ONCOLOGY], **(1999 Nov)** 21 (6) 444-6.
Journal code: 7910681. ISSN: 0253-3766.
CY China
DT Journal; Article; (JOURNAL ARTICLE)
LA Chinese
FS Priority Journals
EM 200202
ED Entered STN: 20020125

Last Updated on STN: 20020208
Entered Medline: 20020207

AB OBJECTIVE: To study the significance of cytokeratin 13 (CK13) gene expression and its methylation in human nasopharyngeal carcinoma (NPC). METHODS: The expression of CK13 in 32 cases of NPC and 8 cases of chronic inflammatory diseases of nasopharyngeal epithelia (CIDNE) was studied using Northern blot hybridization. The **methylation pattern** of CK13 gene was analyzed by Southern blot hybridization using methylation sensitive restriction endonuclease Hpa II and Msp I in NPC cell lines HNE1 and normal human primary cultures of nasopharyngeal epithelial cells. RESULTS: High expression of CK13 gene was found in 8(100%) CIDNE, low-expression of the gene in 12(37.5%) NPC, negative expression in 9(28.1%) and high expression in 11(34.4%). The degree of methylation was increased in NPC **cell** lines HNE1, **compared** to that of normal human primary cultures of nasopharyngeal epithelial cells. CONCLUSION: The expression of the CK13 gene in NPC is partly or completely down regulated. It is possibly related to hyper-methylation of CK13 gene.

L5 ANSWER 2 OF 64 MEDLINE on STN
AN 2000059585 MEDLINE
DN 20059585 PubMed ID: 10590372
TI Evidence that Leydig cells in Sertoli-Leydig cell tumors have a reactive rather than a neoplastic profile.
AU Mooney E E; Man Y G; Bratthauer G L; Tavassoli F A
CS Department of Gynecologic and Breast Pathology, Armed Forces Institute of Pathology, Washington, DC, USA.
SO CANCER, **(1999 Dec 1)** 86 (11) 2312-9.
Journal code: 0374236. ISSN: 0008-543X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200001
ED Entered STN: 20000114
Last Updated on STN: 20000114
Entered Medline: 20000105

AB BACKGROUND: Leydig cells are a variable and an inconstant feature of Sertoli-Leydig cell tumors (SLCT). Controversy exists regarding their neoplastic versus reactive nature, and their molecular biologic profile is unknown. METHODS: Six SLCT and one pure Leydig cell tumor were studied. Mitotic counts and immunohistochemistry for Ki-67 were performed in all cases. Leydig cells, neoplastic tissues, and normal nonneoplastic tissues were microdissected. DNA extracts of these samples were assessed for loss of heterozygosity (LOH) by polymerase chain reaction amplification with ten polymorphic DNA markers that have shown high rates of LOH in a variety of human tumors. Three SLCT and the Leydig cell tumor were assessed for clonality by examining the DNA **methylation pattern** at a polymorphic site on the androgen receptor gene. RESULTS: Leydig cells in SLCT had a low mitotic count (0-1/50 high-power fields [HPF]) compared with the neoplastic stroma (median, 40/50 HPF). Ki-67 was positive in < 2% of Leydig **cells** in all SLCT, **compared** with a median of 7% in the neoplastic stroma. Clonality analysis confirmed the monoclonality of the neoplastic cells in the Leydig cell tumor. However, the Leydig cells from three SLCT were polyclonal, whereas the monoclonal nature of the neoplastic Sertoli tubules was confirmed in one of these cases and that of mucinous heterologous elements in another case. The Leydig cell tumor showed LOH at four of the eight loci evaluated. Leydig cells from five SLCT were evaluated: one showed LOH at one locus, two showed LOH at two loci, and the remaining two showed no LOH. CONCLUSIONS: The demonstration that Leydig cells from SLCT are polyclonal strongly suggests that they are nonneoplastic in nature. This is supported by a low proliferation fraction and a lower fraction of LOH compared with the truly neoplastic Leydig cells.

L5 ANSWER 3 OF 64 MEDLINE on STN
AN 1999392356 MEDLINE
DN 99392356 PubMed ID: 10463060
TI Decreased expression of manganese superoxide dismutase in transformed cells is associated with increased cytosine methylation of the SOD2 gene.
AU Huang Y; He T; Domann F E
CS Department of Radiology, College of Medicine, University of Iowa, Iowa City 52242, USA.
NC CA73612 (NCI)
SO DNA AND CELL BIOLOGY, **(1999 Aug)** 18 (8) 643-52.
Journal code: 9004522. ISSN: 1044-5498.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199909
ED Entered STN: 19990913
Last Updated on STN: 19990913
Entered Medline: 19990902
AB Tumor cells express lower levels of manganese superoxide dismutase (MnSOD) than their normal counterparts. Enforced expression of MnSOD reverses the malignant phenotype of many transformed cells, suggesting that SOD2 is a tumor suppressor. The SOD2 gene contains a large CpG island spanning > 3.5 kb that starts near the 5' edge of the promoter and extends into intron 2. We hypothesized that the difference in SOD2 expression between tumor cells and their normal cell counterparts might be secondary to differences in their cytosine **methylation patterns** in this CpG island. To test this hypothesis, we analyzed the methylation status of the SOD2 gene in two cell line models that show differential MnSOD expression between normal and SV40-transformed cells: WI38 and MRC5 and their SV40-transformed variants, WI38-VA and MRC5-VA. We subdivided the SOD2 gene CpG island into 10 individual regions for analysis by bisulfite genomic sequencing. A region located in intron 2 displayed a significant increase in cytosine methylation in both transformed cell lines that expressed low levels of MnSOD mRNA **compared** with their normal **cell** counterparts. Recent studies by others have shown that SOD2 intron 2 is a potent transcriptional enhancer. The association between increased cytosine methylation of the SOD2 intron 2 region and decreased MnSOD expression in transformed **cells compared** with their normal counterparts suggests that an epigenetic mechanism contributes to the differential SOD2 gene expression between these normal and SV40-transformed cells.

L5 ANSWER 4 OF 64 MEDLINE on STN
AN 1999365304 MEDLINE
DN 99365304 PubMed ID: 10433969
TI Cloning, expression and chromosome locations of the human DNMT3 gene family.
AU Xie S; Wang Z; Okano M; Nogami M; Li Y; He W W; Okumura K; Li E
CS Cardiovascular Research Center, Massachusetts General Hospital, Department of Medicine, Harvard Medical School, Charlestown, MA 02129, USA.
NC GM52106 (NIGMS)
SO GENE, **(1999 Aug 5)** 236 (1) 87-95.
Journal code: 7706761. ISSN: 0378-1119.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF067972; GENBANK-AF135438
EM 199909
ED Entered STN: 19991005
Last Updated on STN: 19991005

Entered Medline: 19990923

AB DNA methylation plays an important role in animal development and gene regulation. In mammals, several genes encoding DNA cytosine methyltransferases have been identified. DNMT1 is constitutively expressed and is required for the maintenance of global methylation after DNA replication. In contrast, the murine Dnmt3 family genes appear to be developmentally regulated and behave like de novo DNA methyltransferases in vitro. In this study, we have cloned human DNMT3A and DNMT3B that encode full-length DNMT3A and DNMT3B proteins with 98% and 94% amino acid sequence identity to their murine homologues. The DNMT3A and DNMT3B show high homology in the carboxy terminal catalytic domain and contain a conserved cysteine-rich region, which shares homology with the X-linked ATRX gene of the SNF2/SWI family. We have mapped human DNMT3A and DNMT3B to chromosomes 2p23 and 20q11.2 respectively, and determined the DNMT3B genomic structure. We further show that DNMT3A expression is ubiquitous and can be readily detected in most adult tissues, whereas DNMT3B is expressed at very low levels in most tissues except testis, thyroid and bone marrow. Significantly, both DNMT3A and DNMT3B expression is elevated in several tumor **cell** lines to levels **comparable** to DNMT1. The cloning of the human DNMT3 genes will facilitate further biochemical and genetic studies of their functions in establishment of DNA **methylation patterns**, regulation of gene expression and tumorigenesis.

L5 ANSWER 5 OF 64 MEDLINE on STN
AN 1999091532 MEDLINE
DN 99091532 PubMed ID: 9872933
TI Reduced levels of poly(ADP-ribosyl)ation result in chromatin compaction and hypermethylation as shown by cell-by-cell computer-assisted quantitative analysis.
AU de Capoa A; Febbo F R; Giovannelli F; Niveleau A; Zardo G; Marenzi S; Caiafa P
CS Department of Genetics and Molecular Biology, University of Rome La Sapienza Rome, Italy.
SO FASEB JOURNAL, **(1999 Jan)** 13 (1) 89-93.
Journal code: 8804484. ISSN: 0892-6638.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199902
ED Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990211

AB The unmethylated status of the CpG islands is important for gene expression of correlated housekeeping genes since it is well known that their methylation inhibits transcription process. An interesting question that has been discussed but not solved is how the CpG islands maintain their characteristic unmethylated status even though they are rich in CpG dinucleotides. Our previous in vitro and in vivo research has shown that poly(ADP-ribosyl)ation is involved in protecting CpG dinucleotides from full methylation in genomic DNA and that a block of poly(ADP-ribosyl)ation is also involved in modifying the **methylation pattern** in the promoter region of Htf9 housekeeping gene. In this study we locked for cytological evidence that in the absence of an active poly(ADP-ribosyl)ation the DNA **methylation pattern** in L929 and NIH/3T3 mouse fibroblast cell lines is altered. For this purpose, differences in the methylation levels of interphase nuclei from control and treated cultures of two murine cell lines preincubated with 2 mM 3-aminobenzamide, an inhibitor of poly(ADP-ribosyl)ation, were measured in individual cells after indirect immunolabeling with anti-5MeC antibodies. The quantitative analysis allowed us to demonstrate that blocking of the poly(ADP-ribosyl)ation results in a higher number, size, and density of antibody binding regions in treated **cells** when

compared to the controls. Analogously, sequential Giemsa staining and indirect immunolabeling of the same slides showed the heterochromatic regions colocalized with the extended methyl-rich domains.

L5 ANSWER 6 OF 64 MEDLINE on STN
AN 1999087337 MEDLINE
DN 99087337 PubMed ID: 9872332
TI Down-regulation of BRCA1 in human sporadic breast cancer; analysis of DNA **methylation patterns** of the putative promoter region.
AU Magdinier F; Ribieras S; Lenoir G M; Frappart L; Dante R
CS Laboratoire de Genetique, UMR 5641 CNRS & avenue Rockefeller, Lyon, France.
SO ONCOGENE, **(1998 Dec 17)** 17 (24) 3169-76.
Journal code: 8711562. ISSN: 0950-9232.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199901
ED Entered STN: 19990209
Last Updated on STN: 19990209
Entered Medline: 19990126
AB Germ-line alterations of BRCA1 are responsible for about 50% of familial breast cancers. Although its biological function(s) has not yet been fully determined, it has been suggested that it may act as a tumor suppressor gene in breast and ovarian cancers. In sporadic breast cancers alterations of BRCA1 have not been detected and in vitro experiments have indicated that BRCA1 negatively regulates cellular proliferation. The present study was designed to identify and quantify, the BRCA1 mRNA levels, in normal and neoplasic human breast tissue. BRCA1 mRNA molecules were quantified using competitive reverse transcriptase PCR assays. DNA **methylation patterns** of this gene have been analysed by Southern blot experiments using methylation sensitive restriction enzymes. We found that BRCA1 mRNA levels were significantly lower in sporadic breast cancers (37 cases analysed, 24 cases of invasive ductal carcinomas not otherwise specified (NOS), two lobular carcinomas in situ two medullary carcinomas, four invasive lobular carcinomas, two invasive mucinous carcinomas and three invasive ductal carcinomas with predominantly in situ component) **compared** with normal breast **tissues** (P=0.0003). This down-regulation of BRCA1 is observed in all histologic types analysed. In invasive ductal carcinomas NOS, this down-regulation does not correlate with any of the prognostic factors studied (tumor size, node status, histologic grade, hormone receptor status). In the samples analysed, alterations of DNA **methylation patterns** were not dectected in the vicinity of the major transcription start site. These data suggest the involvement of BRCA1 in the carcinogenesis of these histologic types.

L5 ANSWER 7 OF 64 MEDLINE on STN
AN 1999027478 MEDLINE
DN 99027478 PubMed ID: 9811339
TI DNA methylation regulates p27kip1 expression in rodent pituitary cell lines.
AU Qian X; Jin L; Kulig E; Lloyd R V
CS Department of Laboratory Medicine and Pathology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota, USA.
NC CA 37231 (NCI)
CA 42951 (NCI)
SO AMERICAN JOURNAL OF PATHOLOGY, **(1998 Nov)** 153 (5) 1475-82.
Journal code: 0370502. ISSN: 0002-9440.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals

EM 199811
ED Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981125
AB We previously reported loss of expression of p27Kip1 (p27) protein in rat GH3 and mouse GHRH-CL1 pituitary tumor **cells compared** with normal pituitary (NP). The molecular basis for the loss of expression of p27 protein in GH3 and GHRH-CL1 cells is unknown. To determine the role of p27 gene methylation in the regulation of the expression of this cell cycle protein, the **methylation patterns** of p27 in normal and neoplastic pituitary cells was analyzed. Inhibition of DNA methyltransferase (DNA-MTase) with 5-aza-2'-deoxycytidine (AZAdC) induced expression of both p27 protein and mRNA when GH3 and GHRH-CL1 cells were treated for 7 days in vitro. DNA methylation correlated inversely with the expression of p27 gene products in NP and pituitary tumor cell lines. Bisulfite genomic sequencing analysis showed that the normally unmethylated cytosines in exon 1 in NP and AtT20 cells were extensively methylated in GH3 and GHRH-CL1 cells. After treatment of GH3 and GHRH-CL1 cells with 10 micromol/L AZAdC, there were decreased numbers of methylated cytosines (by 60% to 90%/o) with variable **methylation patterns** observed by bisulfite genomic sequencing. Analysis of genomic DNA with methylation-sensitive enzymes showed that all SmaI, HhaI, and AvaI enzyme sites of the p27 gene in exon 1 were methylated in GH3 cells but not in NP, confirming the bisulfite genomic sequencing results. AtT20 cells and a human pituitary null cell adenoma cell line (HP75), which expressed abundant p27, had a **methylation pattern** similar to the NP. DNA-MTase activity was elevated fourfold in GH3 cells and twofold in GHRH-CL1 **cells compared** with DNA-MTase activity in NP and AtT20 cells. These results suggest that increased DNA methylation is another mechanism of silencing of the p27 gene in some pituitary tumors and possibly in other types of neoplasms.

L5 ANSWER 8 OF 64 MEDLINE on STN
AN 1999017961 MEDLINE
DN 99017961 PubMed ID: 9799591
TI DNA methylation differences associated with tumor **tissues identified** by genome scanning analysis.
AU Liang G; Salem C E; Yu M C; Nguyen H D; Gonzales F A; Nguyen T T; Nichols P W; Jones P A
CS Department of Biochemistry and Molecular Biology, Urologic Cancer Research Laboratory, Los Angeles, California, 90033, USA.
SO GENOMICS, **(1998 Nov 1)** 53 (3) 260-8.
Journal code: 8800135. ISSN: 0888-7543.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199812
ED Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981231
AB Most investigations on the role of DNA methylation in cancer have focused on epigenetic changes associated with known tumor suppressor genes. This may have led to an underestimation of the number of CpG islands altered by DNA methylation, since it is possible that a subset of unknown genes relevant to cancer development may preferentially be affected by epigenetic rather than genetic means and would not be identified as familial deletions, mutations, or loss of heterozygosity. We used a recently developed screening procedure (methylation-sensitive arbitrarily primed-polymerase chain reaction to scan genomic DNA for CpG islands methylated in white blood cells (WBCs) and in tumor tissues. DNA **methylation pattern** analysis showed little interindividual differences in the WBCs and normal epithelium (adjacent to

colon, bladder, and prostate cancer cells), but with some tissue-specific differences. Cancer cells showed marked methylation changes that varied considerably between different tumors, suggesting variable penetrance of the methylation phenotype in patients. Direct sequencing of 8 of 45 bands altered in these cancers showed that several of them were CpG islands, and 2 of these sequences were identified in GenBank. Surprisingly, three of the bands studied corresponded to transcribed regions of genes. Thus, hypermethylation of CpG islands in cancer cells is not confined to the promoters of growth regulatory genes but is also found in actively transcribed regions.

L5 ANSWER 9 OF 64 MEDLINE on STN
AN 1998449476 MEDLINE
DN 98449476 PubMed ID: 9778046
TI Aberrant methylation of the BRCA1 CpG island promoter is associated with decreased BRCA1 mRNA in sporadic breast cancer cells.
AU Rice J C; Massey-Brown K S; Futscher B W
CS Department of Pharmacology and Toxicology, University of Arizona, Tucson 85721, USA.
NC 3P30 CA23074-19 (NCI)
CA65662 (NCI)
SO ONCOGENE, **(1998 Oct 8)** 17 (14) 1807-12.
Journal code: 8711562. ISSN: 0950-9232.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199811
ED Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981125
AB BRCA1 mRNA is reduced in sporadic breast cancer cells despite the lack of mutations. Because a CpG island is found at the 5' end of the BRCA1 gene, we hypothesized that the decreased BRCA1 mRNA in sporadic breast cancer was associated with aberrant cytosine methylation of the CpG island. We examined BRCA1 mRNA expression in normal human mammary epithelial cells (HMECs), peripheral blood lymphocytes (PBLs) and six sporadic breast cancer cell lines using RT-PCR. The normal breast cells expressed high levels of BRCA1 mRNA. The sporadic breast cancer cell lines and PBLs expressed lower levels of BRCA1 mRNA ranging from a 3-16-fold decrease compared to the normal breast cells. We identified a 600 bp region of the BRCA1 CpG island that possessed strong promoter activity (approximately 40-fold above control), and determined the cytosine **methylation patterns** of the 30 CpG sites within this region by sodium bisulfite genomic sequencing. The HMECs, PBLs and five of the sporadic breast cancer cell lines were largely unmethylated. However, one sporadic breast cancer cell line, UACC3199, was > or = 60% methylated at all 30 CpG sites (18 sites were 100% methylated) and was associated with an eightfold decrease in BRCA1 mRNA **compared** to normal breast **cells** . These findings suggest that aberrant cytosine methylation of the BRCA1 CpG island promoter may be one mechanism of BRCA1 repression in sporadic breast cancer.

L5 ANSWER 10 OF 64 MEDLINE on STN
AN 1998374029 MEDLINE
DN 98374029 PubMed ID: 9710255
TI Variability of DNA **methylation pattern** in somatic and germ cells in male newt (Amphibia, Urodela) Triturus cristatus carnifex.
AU Pontecorvo G; De Felice B; Carfagna M
CS Faculty of Biological Science, Department of Life Sciences, II University of Naples, Caserta, Italy.
SO FEBS LETTERS, **(1998 Jul 31)** 432 (1-2) 77-81.
Journal code: 0155157. ISSN: 0014-5793.

CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199809
ED Entered STN: 19980917
Last Updated on STN: 19980917
Entered Medline: 19980910
AB In a survey of several mammalian genomes, namely humans, rodents and bovines, the differences in the 5-methylcytosine (m5C) content show that repeated DNA sequences from sperm were undermethylated and from various somatic tissues were heavily methylated. This report shows a **pattern** of **methylation** in male newt (Amphibia, Urodela) Triturus cristatus carnifex (T. c. c.) unlike that so far described by other authors in mammals. Using methylation sensitive and insensitive enzymes (HpaII and MspI) and successive 3' terminal labelling (fill-in), we found a greater degree of DNA methylation in premeiotic germ and sperm **cells compared** to somatic **tissue** such as hepatocytes. Furthermore the degree of total DNA methylation in spermatozoa appears somewhere between premeiotic germ cells and somatic tissue. Blot hybridization shows that two highly conserved repetitive sequences in amphibian T. c. c., pTvm1 and pTvm8, contribute significantly to the degree of DNA methylation, suggesting a function for these sequences, such as a role in transcriptional regulation.

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FULL ESTIMATED COST	18.27	18.48

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